Vacuum-assisted apical suction devices induce passive electrical changes consistent with myocardial ischemia during off-pump coronary artery bypass graft surgery

Roger Dzwonczyk,*, Carlos L. del Rio, Chittoor Sai-Sudhakar, John H. Sirak, Robert E. Michler, Benjamin Sun, Nicole Kelbick, Michael B. Howie

*Department of Anesthesiology, The Ohio State University, Columbus, OH 43210, USA
bDepartment of Electrical & Computer Engineering, The Ohio State University, Columbus, OH 43210, USA
cDivision of Cardiothoracic Surgery, The Ohio State University, Columbus, OH 43210, USA
dCenter for Biostatistics, The Ohio State University, Columbus, OH 43210, USA
eDepartment of Cardiothoracic Surgery, Montefiore Medical Center, Bronx, NY, USA

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Abstract

Objective: Off-pump coronary artery bypass graft surgery is common therapy to completely revascularize diseased hearts. In order to graft posterior arteries in this procedure, the heart must be lifted from the chest cavity and manipulated to expose the surgical field using an apical suction device. This suction device may cause unwanted myocardial ischemia.

Methods: In this observational study, we measured myocardial electrical impedance, a parameter that responds to myocardial ischemia, as well as ST-segment changes during off-pump coronary artery bypass graft surgery in 12 patients with two-vessel coronary artery disease undergoing revascularisation of the left anterior descending and the posterior descending coronary arteries. During the posterior descending artery revascularisation phase of the procedure the apical suction device was oriented over the electrodes used to measure myocardial electrical impedance, thus allowing us the opportunity to assess myocardial ischemia in this region of the heart.

Results: In these 12 patients, myocardial electrical impedance progressively increased under the suction device during posterior coronary artery revascularisation, suggesting that myocardial ischemia developed in this region of the myocardium. ST-segment changes were negligible while the heart was vertically displaced (and the suction device attached), but increased immediately when the heart was returned to the neutral anatomical position.

Conclusion: Our data suggest that the apical suction device may cause ischemia while the heart is vertically displaced and electrically disconnected from the body. Under these conditions, ST-segment changes may not detect myocardial ischemia. Myocardial electrical impedance has the potential to reliably detect intraoperative myocardial ischemia under these circumstances.

Keywords: Myocardial electrical impedance; Apical suction devices; Off-pump coronary artery bypass surgery; Myocardial ischemia

1. Introduction

Despite significant improvements in angiographic and therapeutic techniques, coronary artery bypass graft (CABG) surgery remains the gold standard for complete myocardial revascularisation [1]. Such status has been aided by the development of its off-pump (OP) variant or OPCABG surgery, which involves revascularisation while the heart continues to beat and circulate blood systemically. For example, Raja and Dreyfus [2] recently pointed out the potential benefits of OPCABG surgery, over traditional on-pump CABG surgery, particularly in older, high-risk patients. Off-pump CABG surgery is a far less complicated procedure than traditional CABG surgery and produces less intraoperative and post-operative complications in patients with comorbidity and/or more advanced atherosclerotic disease.

However, there are caveats. Initially reserved for grafting left anterior descending coronary arteries (LADa), OPCABG surgery has been extended to multi-vessel disease involving posterior coronary arteries. For this purpose, several vacuum-assisted apical suction devices have been developed; these devices expose the posterior descending coronary arteries (PDA) and help stabilize the surgical field. The UrchinTM (Medtronic Corporation, Minneapolis, MN, USA) and the XposeTM (Guidant Corporation, Santa Clara, CA, USA) are two examples of apical suction devices used in OPCABG surgery today. Although definitive evidence is lacking, the literature suggests that these devices may inadvertently cause significant myocardial ischemia in the contact region of
the heart [3], potentially causing ischemic injury and therefore diminish the benefits of OPCABG surgery. Fernández et al. [4] recently demonstrated that the apical suction device causes a decrease in the partial pressure of myocardial oxygen, and concluded that such devices cause severe myocardial ischemia of the suctioned myocardium.

In order to shed further light on this subject, we present here our observations on myocardial electrical impedance (MEI) in patients undergoing CABG revascularisation of the PDa and compare MEI changes with ST-segment changes, which are often considered the benchmark for myocardial ischemia. Myocardial electrical impedance has been found to increase predictably with regional myocardial ischemia in humans and animals [5,6]. This electrical impedance increase has been well correlated with the functional [7], ionic, and metabolic state of the myocardium [8], and hence, should reflect any ischemic insult induced by an apical suction device.

2. Materials and methods

After obtaining FDA and institutional approval as well as informed written patient consent, we recorded MEI and ST-segment changes (2 leads, automatic trending) [9], in 12 patients with angiographically-demonstrated two-vessel disease undergoing OPCABG surgery of the left anterior and the posterior descending coronary arteries.

The subjects enrolled in this study had a severely stenosed PDa (80—100%) and grafting to that vessel was achieved with the aid of the Medtronic UrchinTM apical suction device. These patients are a subset of those enrolled in our overall MEI studies during LADa revascularisation [5,6] and were selected because the apical suction device was attached directly over the MEI electrodes. The MEI of the tissue under the suction device was measured during PDa and LADa revascularisation, i.e., with and without the suction device attached and functioning. Two time-points — pre-occlusion and occlusion — were observed during the LADa revascularisation, and three — pre-UrchinTM attachment, UrchinTM attached and post-UrchinTM attachment — were recorded during the PDa grafting. For purposes of this report, each time-point is defined as the 30 s ensemble-average of MEI and ST-segment values. No coronary shunts were used in the surgical procedure. Vascular loops were used as needed to control bleeding. In these cases, the tissue at the surgical site was stabilized via a Medtronic OctousTM tissue stabilizer. The surgeons utilized a blower/mister system (such as the Medtronic AccuMistTM) to aid in the visualization of the surgical site.

Myocardial electrical impedance was recorded in a fashion previously described in detail [5]. Our MEI monitor consists of a laptop computer that communicates with and controls custom analog circuitry that, in turn, connects to the heart via two temporary pacing electrodes (MYOWIRE size 2-0 [3.0 metric] temporary cardiac pacing wires, A&E Medical Corporation, Farmingdale, NJ, USA) attached to the myocardium approximately 1 cm apart in the distal LADa distribution. A 5-μA 100 μs impulse is impressed on the myocardium through the temporary pacing electrodes and the current impulse and resultant voltage drop across the myocardial tissue between the electrodes are measured, amplified, filtered and digitized at 22.0 kHz and 12-bit resolution, and then transformed into the frequency domain via fast Fourier transformation. Myocardial electrical impedance is calculated by dividing the associated voltage and current spectra at each frequency interval and then averaging over the resulting impedance spectrum. Our spectrum ranges from 0.5 kHz to 5.0 kHz and the frequency domain resolution is 5.3711 Hz in that range.

3. Statistical analysis

Data are presented as mean ± standard deviation (SD) and summarized in Table 1. Although MEI measurement uncertainty is dominated by Gaussian (normally distributed) noise [10], the data were collected under different experimental circumstances; thus, factors other than measurement noise (electrode location, surgical preparation, surgeon, etc.) may have affected the measurement process, and normality assumptions may not be appropriate. Therefore, MEI and ST-segment changes during LADa (pre-/during occlusion) and PDa (pre-/during/post-UrchinTM attachment) revascularisation were analysed using the non-parametric Wilcoxon signed-rank test. Statistical significance was set a priori at p < 0.05, and adjusted for multiplicity via the Bonferroni’s method. As previously reported, absolute ST-segment deviations >1 mm were considered as clinically indicative of ischemia [11,12]. In addition, multiple linear regression

<table>
<thead>
<tr>
<th>Time point</th>
<th>ST-segment (mm) (N = 10)</th>
<th>MEI (Ω) (N = 12)</th>
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<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>p-value</td>
</tr>
<tr>
<td>LADa</td>
<td></td>
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<tr>
<td>Pre-occlusion</td>
<td>0.183 ± 0.181</td>
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<tr>
<td>Occlusion</td>
<td>0.332 ± 0.578</td>
<td>0.0137</td>
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<tr>
<td>UrchinTM</td>
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<tr>
<td>Pre-attachment</td>
<td>0.203 ± 0.144</td>
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<tr>
<td>Attached</td>
<td>0.168 ± 0.125</td>
<td>0.0000</td>
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<tr>
<td>Post-attachment</td>
<td>1.802 ± 1.081</td>
<td>0.0006</td>
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<tr>
<td>Pre versus post</td>
<td>0.0060</td>
<td>0.0368</td>
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</tbody>
</table>

* p-values are based on the Wilcoxon signed-rank test and are adjusted for multiplicity in the Urchin analyses; results compare the current time point to the previous time point unless otherwise noted.

* p-value is based on comparisons between pre- and post-attachment results.
4. Results

Myocardial electrical impedance data were collected from all 12 patients. ST-segment data were collected from 10 of the 12 patients. As expected [4,5], MEI increased immediately with LADa occlusion (+9.46 ± 5.31%, \( p < 0.05 \)), and was accompanied by a significant but moderate (<1 mm) ST-segment deviation (0.532 ± 0.578 mm) (see Fig. 1 and Table 1).

Similar MEI changes (+19.65 ± 7.62%) were observed during PDA exposure with the apical suction device, i.e., heart vertically displaced (without occlusion), suggesting that myocardial ischemia developed under the UrchinTM. In our cases, MEI began increasing as soon as the suction device was attached to the heart and suction was applied. Our data point — UrchinTM attached — is an average value that includes MEI measurements during both device attachment and vertical lifting of the heart since our surgeons attach the device and manipulate the heart in rapid sequence. While the heart was vertically displaced (suction device attached) negligible ST-segment deviations were recorded (see Table 1, UrchinTM attached). However, immediately after the heart was returned to the neutral position (post-UrchinTM), significant ST-segment changes were present (1.802 ± 1.081 mm) and three patients developed ventricular fibrillation (successfully cardioverted to normal sinus rhythm), strongly suggesting that, in fact, myocardial ischemia was present. The results of the multiple linear regression analysis are summarized in Fig. 2, where early (first 2.5 min) MEI changes during UrchinTM attachment are shown to occur faster (0.95%/min, \( R^2 = 0.941, p < 0.001 \)) than after complete LADa occlusion.

5. Discussion

This study offers further evidence suggesting that apical suction devices, used in OPCABG surgery for cardiac positioning, cause myocardial ischemia. We observed that the attachment of one such device produced immediate MEI changes similar to those recorded during complete coronary artery occlusion (Figs. 1 and 2). This early MEI time course has been well demonstrated to reflect myocardial ischemia [7,5,6]. Thus, our results are in good agreement with the initial report of D’Ancona et al. [3], who observed sudden hemodynamic impairment, decreased graft flow and ischemic ST-segment elevation associated with the use of an apical suction device during an OPCABG procedure.

While the etiology of this ischemic insult is not clear; D’Ancona et al. [3] hypothesized that apical suction devices could partially compress the distal segment of a native coronary artery, limiting flow (both native and grafted) and causing regional myocardial ischemia. We have recently shown that a faster MEI time course following acute coronary occlusion is suggestive of reduced collaterals, and hence, a more severe ischemia [6]. Thus, while similar MEI changes after LADa occlusion and UrchinTM attachment supports the coronary compression theory, the higher slope of the latter is also suggestive of reduced microcirculation under the apical suction device. Such additional ischemia could result from the negative pressure exerted by the positioning device over the collateral-dependant (due to coronary compression) myocardial tissue. However, the original work on suction stabilization [13] reported no ill effects of negative suction applied directly over a coronary artery. Hence, reduced regional microperfusion is more likely a direct consequence of collateral closure due to impaired ventricular hemodynamics during heart displacement with the vacuum-assisted apical suction device [14,15].

In addition, and perhaps more importantly, our results expose a fundamental limitation of traditional ischemic indexes (e.g., ECG ST-segment trending, echocardiographic wall-motion) during beating-heart surgical revascularisation procedures, that being their dependence on the degree of
coupling (electrical, acoustic, etc.) between the myocardium and the pericardial well. For example, when the heart is vertically displaced from the chest cavity to expose the posterior surgical field (e.g., during PDa revascularisation), the electric circuit between the ECG generator — the heart — and the peripheral ECG electrodes is disrupted. As a result ST-segment changes, and in general any ECG-derived ischemic index, becomes unreliable during OPCABG surgery, and perhaps, should not be the method of choice by the cardiothoracic surgeons/anesthesiologists to assess intraoperative acute myocardial ischemia. Therefore our observations suggest the need for more reliable and sensitive methods to detect intraoperative myocardial ischemia, such as MEI.

6. Conclusion

With the use of MEI monitoring, we have provided further evidence here that apical suction devices used to expose and stabilize the posterior surgical field during OPCABG revascularisation may cause myocardial ischemia, a condition undetected by ECG ST-segment trending analyses. Thus, this study highlights the need for a more reliable and sensitive method for detecting myocardial ischemia during OPCABG surgery. MEI has the potential to monitor intraoperative myocardial ischemia in this instance.

References